

Editorials

Indoor Radon—What Is to Be Done?

ELSEWHERE IN THIS ISSUE of the journal, Samet reviews some of the current approaches for assessing indoor radon exposure and the associated risks for developing lung cancer.¹ It is not surprising that this topic is received with considerable interest. If, as he concludes, 10,000 to 20,000 cases a year of lung cancer result from indoor radon exposure, this is certainly a serious ecologic problem. If true, the significance of this assertion is critically dependent on the degree to which the radon-induced cases are preventable. If they were all preventable, the potential effects on lung cancer mortality would be dramatic. Because the overall five-year survival rate for lung cancer is only about 10%, eliminating all radon-induced lung cancer would be comparable to a 100% increase in the theoretical five-year survival (calculated by comparing the five-year survival of a population that includes persons with exposure to radon and a similar population in which the radon exposure has been eliminated).² Unfortunately, as with most things in life, things are not as simple as they might seem.

In this review, a number of assumptions are made that appear to be reasonable and straightforward on the surface but may leave readers with a distorted perspective of what the practical implications are. Extrapolating indoor radon risk from that of radon exposure in miners is generally based on the assumption that there is a linear-no threshold relationship between radiation exposure (radon) and carcinogenesis (induction of lung cancer). Implicit in this theory is the dose-rate independence of the biologic effects of radiation. Several recent studies provide convincing evidence that the relative risk of radon-induced lung cancer is, in fact, not independent of the dose rate.

Hornung and Meinhardt, for example, found that among a cohort of uranium miners, low exposure rates were more harmful per unit of cumulative exposure than high-dose rates.³ Similar findings were more recently reported by Lubin and co-workers and supported in an analysis by Darby and Doll.^{4,5} A formal test of the linear-no threshold theory of carcinogenesis was reported by Cohen⁶ who, using data from 411 counties from various parts of the United States, reported a negative correlation between radon exposure and the risk of developing lung cancer. The failure to confirm the validity of the linear-no threshold theory in this study may reflect the fact that low-rate radon irradiation is a more efficient inducer of lung cancer. If the dose rate is as important a factor as the total dose is for predicting cancer risk, an analysis that compares risk by dose (ignoring dose rate) might find a negative correlation. In any event, the available data suggest that the relationship between radon exposure and subsequent lung cancer risk is complex and not simply linear.

The significance of these dose-rate effects has major implications, particularly in light of the distribution of rates of exposure in the general population. Radon exposure rates in single-family homes follow a log-normal distribution skewed so that the vast majority of homes have low exposure rates.⁷ Because low-dose rate exposure is more efficient at inducing lung cancer and because the vast majority of households have low rates of exposure, it follows that the vast majority of radon-induced lung cancers probably result from relatively

low-dose rate exposures. Attempting to remedy this problem by identifying the small percentage of persons with exposure to relatively high-dose rates is not likely to be cost effective nor to result in a substantial reduction in lung cancer-related mortality.⁸ The cost of a national effort to uniformly reduce indoor radon exposure could be considerable. For example, if, as proposed by some, the indoor radon levels were reduced to those found in outside air, the estimated average cost to homeowners would be approximately \$10,000 each.⁹

The most cost-effective way to reduce the incidence of radon-induced lung cancer appears to be to reduce the number of smokers (there appears to be some synergism between smoking and radon exposure).^{9,10} Additional measures that might be considered include improving the ventilation systems in existing homes (particularly in parts of the country where risk has been identified) and modifying the requirements for the ventilation systems for new constructions. Educating the population to do simple things, such as avoiding the use of basements as major living areas and discouraging the improper handling of actinide-containing mineral collections displayed in living areas, are further examples of potentially cost-effective intervention.¹¹ If funds that could be spent on smoking cessation programs are diverted to reduce radon exposure, it is possible that the lung cancer rates might actually increase!

Samet's review of current approaches for assessing the risk of indoor radon exposure brings the issue to mind but raises more questions than it answers. As he suggests, despite some uncertainty, data obtained from radon exposure in miners appear to have provided valuable information for assessing the indoor risk. But in light of these uncertainties, what is to be done? Until proved otherwise, it appears that the most cost-effective way to reduce radon-related lung cancer mortality is to discourage smoking, to encourage good indoor ventilation, and to discourage other behaviors that are likely to result in higher exposures to radon gases.

MACK ROACH III, MD
Assistant Professor, Department of
Radiation Oncology

KEITH A. WEAVER, PhD
Associate Professor, Department of
Radiation Oncology
University of California, San Francisco
School of Medicine
San Francisco, California

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